and disseminated herpes infections in newborn infants.

Interferon, a natural body protein, has been produced in vitro in quantities sufficient for administration to patients and has been found effective against herpes zoster. However, it remains both difficult and expensive to produce.

In addition to the parenteral drugs mentioned, idoxuridine has long been an effective topical therapy for treatment of certain forms of herpetic keratitis, and topical adenine arabinoside appears even more beneficial in ocular disease.

Newer agents with considerable promise are being tested. They include acycloguanosine (Acyclovir), which is much more effective in vitro than adenine arabinoside against herpes viruses, and the glucose derivative 2-deoxy-D-glucose, which has been reported to be beneficial for genital herpes infections when applied topically.

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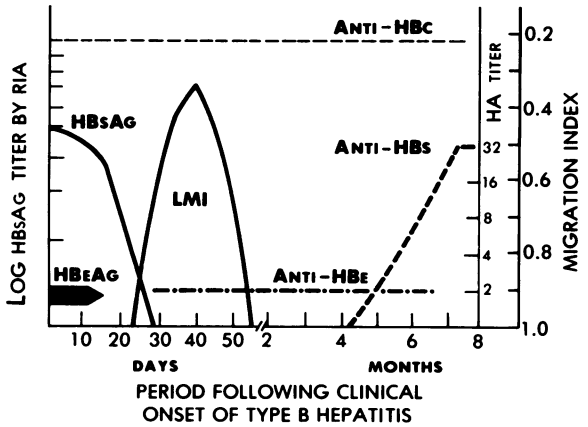
Immunodiagnosis of Viral Hepatitis

LABORATORY DIAGNOSIS OF HEPATITIS types A, B and non-A/non-B (abbreviated HA, HB and NANB, respectively) can be established by testing immunologic responses to the viral agents of HA and HB; NANB is established only by exclusion (Table 1).

In HA, the presence of anti-HA in the IgM class of immunoglobulins suggests a current or recent infection, whereas anti-HA appearing in the IgG class indicates infection occurring in the past and confers immunity to HA.



Anti-HBc = hepatitis B core antibody; Anti-HBe = hepatitis B "e" antibody; Anti-HBs = hepatitis B surface antibody; HBeAg = hepatitis B "e" antigen; HBsAg = hepatitis B surface antigen; RIA = radioimmunoassay.

Figure 1.—Immunologic response to HBsAg, HBcAg and HBeAg during acute and convalescent phases of type B hepatitis. The fact that leukocyte migration inhibition (LMI) with purified HBsAg is present but disappears after about four weeks suggests that hepatitis B virus infection may be self-limited. In contrast, the LMI test is negative in persistent or chronic cases of hepatitis B.

The temporal changes in various markers during the acute phase of HB are shown in Figure 1. Although the clinical manifestations of acute HA, HB or NANB are similar, a chronic carrier state and chronic liver disease of more than six months duration are recognized only for infection with HB or NANB agents. Nearly 10 percent of patients with acute HB have unresolved hepatitis, frequently persisting for years, and often progressing to a

TABLE 1.—*Practical Guide for the Interpretation of Serologic Markers of Viral Hepatitis*

Clinical Interpretation	*IgM Anti-HA**	*HBsAg*	*HBeAg*	*Anti-HBe*	*Anti-HBc*	*Anti-HBs*
Acute HA	+	−	−	−	−	−
Incubation period or early acute HB	−	+	+	−	−	−
Acute HB	−	+	+	−	+	−
Fulminant HB	−	+	−	−	+	+/−
Convalescence from acute HB	−	−	−	+	+	+/−
Chronic HB	−	+	+/−	+/−	+	+/−
Persistent HB carrier state	−	+	−	+	+	−
Past infection with HB virus	−	−	−	−	+	+
Infection with HB virus without detectable (excess) HBsAg	−	−	−	−	+	−
Immunization without infection	−	−	−	−	−	+
Non-A/non-B hepatitis by exclusion of markers for HA and HB	−	−	−	−	−	−

Anti-HA = hepatitis A antibody; HBsAg = hepatitis B surface antigen; HBeAG = hepatitis B "e" antigen; anti-HBe = hepatits B "e" antibody; anti-HBc = hepatitis B core antibody; anti-HBs = hepatitis B surface antibody.

*Clinically unrecognized infection occurring in childhood accounts for the high incidence of IgG anti-HA antibodies in 80 percent to 90 percent of adults in underdeveloped countries as compared with 25 percent to 50 percent of adults in Europe and the United States.

form of chronic hepatitis. The pathogenic mechanisms responsible for the persistence of viral infection and the chronic hepatic condition are unclear.

Asymptomatic carriers of HB, estimated to number about 900,000 in the United States, and possibly 150 million worldwide, are believed to serve as an epidemiologic reservoir for HB virus infection. Because a large percentage of patients with primary hepatocellular carcinoma are positive for the hepatitis B surface antigen (HBsAg), HB virus is suspected to be oncogenic. Because the hepatitis B surface antibody (anti-HBs) is protective against reinfection with HB virus, HBsAg purified from the plasma of chronic carriers is being evaluated as a prophylactic vaccine against HB and its sequelae.

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